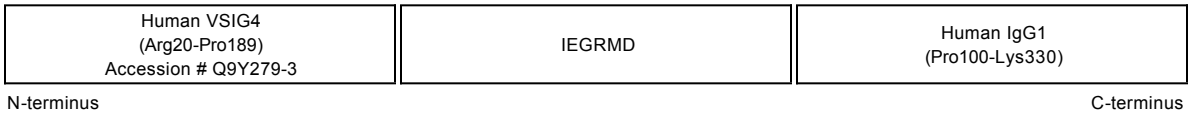
Recombinant Human VSIG4 Short Isoform Fc Chimera

Catalog Number: 9817-VS

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived



N-terminal Sequence Analysis	Arg20
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	46 kDa

SPECIFICATIONS

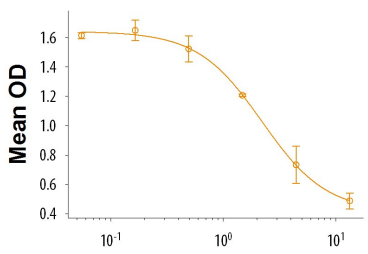
SDS-PAGE	55-65 kDa, reducing conditions
Activity	Measured by its ability to inhibit anti-CD3 antibody induced IFN-gamma secretion by human peripheral blood mononuclear cells (PBMC). The ED_{50} for this effect is 1-5 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

Bioactivity



Recombinant Human VSIG4 Fc Chimera (Catalog # 9817-VS) inhibits IFN-γ secretion by human peripheral blood mononuclear cells in the presence of anti-CD3 antibody. The ED_{50} for this effect is 1-5 µg/mL.

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BACKGROUND

VSIG4 (V-set and immunoglobulin domain containing 4), also known as CRIg and Z39IG, is a type I transmembrane protein of the B7 family within the Ig superfamily that is expressed only in tissue-resident macrophages (1-4). The gene is located on the X chromosome (2). The human VSIG4 precursor includes a signal sequence, an extracellular domain (ECD) containing a V-type and a C2-type Ig domain, a transmembrane domain and a cytoplasmic domain (3). Splice isoforms lacking all or part of the cytoplasmic domain, the C2-type Ig domain and/or the transmembrane domain have been identified (5). This product is a VSIG4 isoform lacking C2-type Ig domain (Short Isoform). The human VSIG4 ECD shares 84% aa identity with canine VSIG4. Within the IgV domain, it shares 90%, 80% and 78% aa identity with bovine, mouse and rat VSIG4, respectively; these animals lack the C2-type domain. VSIG4 is specifically expressed on macrophages in the thymic medulla, peritoneum, alveoli, synovia, adipose and heart, liver Kupffer cells, placental Hofbauer cells, and atherosclerotic foam cells (1-4, 6-9). It is absent on infiltrating macrophages (8). VSIG4 is a complement receptor that binds C3b and iC3b fragments, internalizes them to recycling endosomes, and is recycled to the cell surface (4, 6). It contributes significantly to innate immunity by binding and phagocytosis of complement-opsonized invading pathogens (4, 8, 10). Binding of either native or recombinant soluble VSIG4 to C3b inhibits complement amplification through the alternative, but not classical, pathway (10, 11). VSIG4 is also a negative regulator of mouse and human T cell activation (2). Although VSIG4 engagement may activate NF kappa B and thus be pro-inflammatory in some cases, many of its activities are important in resolving, rather than initiating, inflammation (1, 2, 7, 10, 11). VSIG4 negatively regulates macrophage activation by reprogramming mitochondrial pyruvate metabolism (12). VSIG4 is overexpressed in ovarian cancers compared with that in benign tumors (13). VSIG4 expression in multiple myeloma is an independent indicator of poor prognosis, implying a possible therapeutic target for immunotherapy for multiple myeloma (14).

References:

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